Chronic Beryllium Disease and Cancer Risk Estimates with Uncertainty for Beryllium Released to the Air from the Rocky Flats Plant

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Beryllium was released into the air from routine operations and three accidental fires at the Rocky Flats Plant (RFP) in Colorado from 1958 to 1989. We evaluated environmental monitoring data and developed estimates of airborne concentrations and their uncertainties and calculated lifetime cancer risks and risks of chronic beryllium disease to hypothetical receptors. This article discusses exposure–response relationships for lung cancer and chronic beryllium disease. We assigned a distribution to cancer slope factor values based on the relative risk estimates from an occupational epidemiologic study used by the U.S. Environmental Protection Agency (EPA) to determine the slope factors. We used the regional atmospheric transport code for Hanford emission tracking atmospheric transport model for exposure calculations because it is particularly well suited for long-term annual-average dispersion estimates and it incorporates spatially varying meteorologic and environmental parameters. We accounted for model prediction uncertainty by using several multiplicative stochastic correction factors that accounted for uncertainty in the dispersion estimate, the meteorology, deposition, and plume depletion. We used Monte Carlo techniques to propagate model prediction uncertainty through to the final risk calculations. We developed nine exposure scenarios of hypothetical but typical residents of the RFP area to consider the lifestyle, time spent outdoors, location, age, and sex of people who may have been exposed. We determined geometric mean incremental lifetime cancer incidence risk estimates for beryllium inhalation for each scenario. The risk estimates were < 10^{-6}. Predicted air concentrations were well below the current reference concentration derived by the EPA for beryllium sensitization. *Key words*: atmospheric transport modeling, beryllium, chronic beryllium disease, exposure assessment, lung cancer, uncertainty. *Environ Health Perspect* 107:731–744 (1999). [Online 3 August 1999]
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The Rocky Flats Environmental Technology Site in Colorado is owned by the U.S. Department of Energy. For most of its history, the site was called the Rocky Flats Plant (RFP) and was operated as a nuclear weapons research, development, and production complex. The RFP is located on approximately 2,650 ha (6,500 acres) of federal property about 8–10 km (5–6.2 miles) from the cities of Arvada, Westminster, and Broomfield, Colorado, and 26 km (16 miles) northwest of downtown Denver, Colorado. The original 156-ha (385-acre) main production area is surrounded by a 2,490-ha (6,150-acre) buffer zone that now delineates the RFP boundary.

In this paper we describe risk calculations performed to estimate inhalation of beryllium resulting from operational and accidental releases at the RFP. We evaluated soil and sediment monitoring data for beryllium and studied evidence of carcinogenicity and chronic beryllium disease. We also describe environmental transport modeling, provide estimates of uncertainty in the model predictions, and present distributions of carcinogenic risk resulting from the inhalation of beryllium for several generic receptor scenarios.

Beryllium Release Estimates

Beryllium was initially used in research and development in 1953. Beryllium operations became significant from 1958 to 1975 at the RFP. The details of beryllium component manufacturing, machining, cutting, heat treating, rolling, and other operations and ventilation systems used to control beryllium emissions over the years are described in technical reports by ChemRisk (*1*) and McGavran et al. (*2*) and in a letter written by Campbell (*3*). With the possible exception of effluent from one building in the early 1960s, all air exhaust discharged from RFP beryllium-processing facilities was subjected to high-efficiency particulate air (HEPA) filtration to control radioactive effluents (*4*).

Beryllium has been monitored in the plant air exhaust effluent since at least 1963 (*1,4,5*). The monitoring program data for routine airborne emissions of beryllium provided the basis for the release estimates shown in Figure 1. Beryllium emissions were determined from sample data log books for 1960–1970 and from annual beryllium releases reported in the annual environmental monitoring reports for 1971–1989. The log books contain daily sample results for workroom air and building effluents. ChemRisk (*4*) calculated the monthly and annual average beryllium concentrations for each stack from the building effluent data. Because data on exhaust flow rates and total exhaust volume were lacking for some facilities, releases were estimated using facilities of similar size (*4*). No sampling data from before 1960 were located. Therefore, we assumed that emissions in 1958 and 1959 were the same as those reported in 1960.

Air exhaust samples were taken from filter plenum exhausts after the air passed through HEPA filters but before it exited the stack. The sampling practices, sampling system design, sample line losses, calculations of flow rates, and exhaust volume and uncertainties determined previously for radioactive particles were applied to the beryllium sampling data (*4*).

Beryllium was also released during three fires that occurred in 1962, 1964, and 1978 (*6,7*). These releases were monitored by the stack sampling equipment; therefore, they were included in the yearly release estimates (*4*). The most significant fire occurred on 23 February 1978. A release estimate of 14.5 g from the fire was included in the < 17-g release estimate for 1978. The 1978 release estimate was based on monitoring results from the plenum sampler, ambient air sampling, and samples of water used to fight the fire. The water that was used to fight the fire drained into and was sampled from ponds, ditches, and temporary impoundments (*4,8–10*).

Release estimates typically ranged from 10 to 30 g/year for the years 1958–1971 and generally were < 10 g/year after 1971. Documentation suggests that beryllium measurement data handling practices may have led to reporting annual emissions that were greater than actual releases (*4*). Beryllium releases from 1971 to 1989 were obtained from the annual environmental monitoring reports issued by the RFP, which often reported beryllium release totals for the year as less-than values. The 1975 report (*11*) explained that samples with less than the minimum detectable concentration

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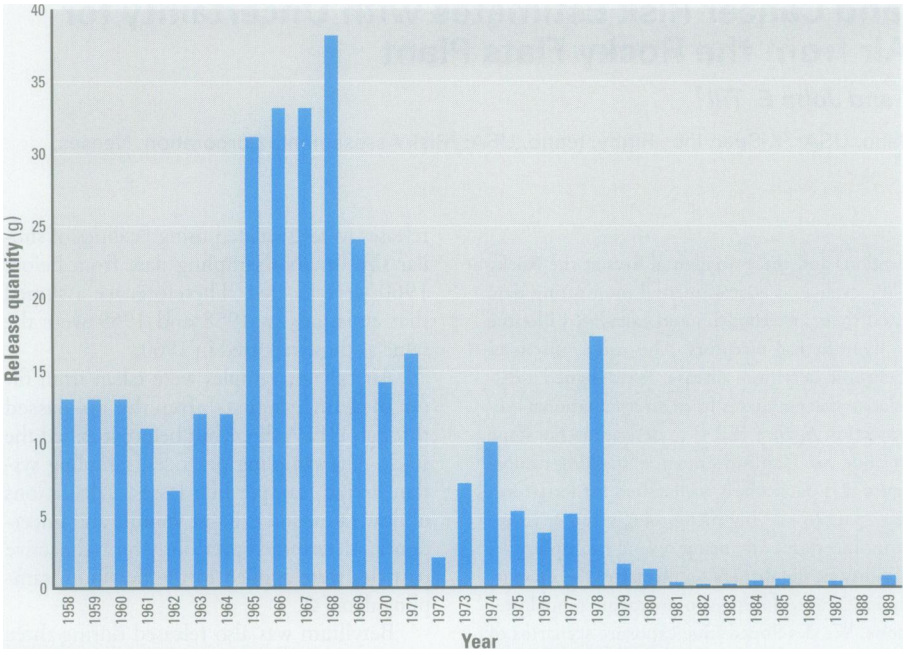

Figure 1. Annual release estimates for beryllium, as estimated by ChemRisk (*4*).

were considered to be at the minimum detectable concentration for averaging. Using the minimum detectable amount value for beryllium at each effluent measurement location resulted in a calculated minimum beryllium discharge of 0.4 g/month (*12*). The annual report estimated that the total release from 1958 to 1970 was 253.7 g. The total release from 1971 to 1989 was 70.5 g; therefore, the total release from 1958 to 1989 was 324 g.

The uncertainty associated with the beryllium source term estimates was characterized. Uncertainties in exhaust and sample flow rate estimates and in analytical results were combined, and the total uncertainty was calculated using Monte Carlo methods.

Environmental Monitoring for Beryllium

Surface water. Beryllium was transferred offsite in creeks that flowed to surface water that was used for drinking. This exposure pathway has been well characterized because of public concerns about tritium releases to surface water. Beryllium has been monitored in water effluent since 1980 (*4*). Routine surface water monitoring has always shown < 0.05 mg beryllium per liter of water, which is the analytical detection limit.

The beryllium compounds of concern are not very water soluble and would be expected to bind to sediments and soils. Beryllium concentrations in the sediments of two lakes near the plant, Great Western Reservoir and Standley Lake, are similar to background levels and concentrations found in soil and sediment samples from other Rocky Mountain regions (*4,13,14*).

Historically, inhalation of beryllium has been a greater human health concern than ingestion, in part because < 1% of ingested beryllium is absorbed through the gastrointestinal tract (*15*). Beryllium does not bioaccumulate in fish. We did not further evaluate releases of beryllium to surface water because of a lack of source term and effluent and environmental monitoring data, insufficient evidence of accumulation in soils and sediments, and the low solubility and gastrointestinal absorption of beryllium.

Soil. Beryllium concentrations in soil are of interest because beryllium can be resuspended in soil and a pattern of beryllium contamination in soil could reveal information about discharges from the plant. Beryllium sources that might affect concentrations in soils at the RFP include operations at the plant, a beryllium ore industry located 2 km (1.2 miles) east of the plant, a beryllium ceramics industry 15 km (9.3 miles) south of the plant, beryllium in gravel brought into the site, coal burning and other combustion sources near the plant, and naturally occurring beryllium (*16,17*).

In 1982, a site study was conducted to characterize sources of beryllium and to determine beryllium concentrations in soil (*16*). This study estimated that 196 g beryllium was exhausted from all buildings which processed beryllium during the 24 years from 1958 to 1982. For the study, researchers gathered 241 soil and rock samples from the site and nearby areas. Deeper samples were taken at 5–10 cm (2–4 inches) to establish the geological background levels of beryllium. The study concluded that RFP-originated beryllium could not be distinguished from geological naturally occurring beryllium taken from land outside plant property. Higher levels found near roads and buildings were attributed to surficial gravel aggregates. The survey found that the natural gravels and 36 million kg of gravel brought in and added to RFP surfaces had the highest and most variable beryllium concentrations. The mean concentration in these gravels was 1.1 ± 1.4 µg/g soil (parts per million or milligrams per kilogram of soil). The background beryllium concentrations in soil (Rocky Flats alluvium) averaged 0.64 ± 0.07 µg/g. The mean level in soils in the plant area was 0.6 µg/g and ranged from 0.2 to 1.1 µg/g.

The Barrick study (*16*) suggested that atmospheric transport of beryllium to soils surrounding the plant had not occurred because surficial soils near the plant did not have elevated beryllium concentrations.

The Colorado Department of Public Health and Environment (Denver, CO) conducted studies on beryllium in soil in 1971 and 1989. The 1989 study reported 21 results, all less than the analytical detection limit of 2.7 µg/g. The 1971 data consisted of 13 results, ranging from 2.0 to 60 µg/g, with no analytical detection limit reported. The pattern of positive values seen in 1971 was not consistent with the pattern expected if the beryllium in the soils had been deposited by atmospheric dispersion from the RFP. Spatial variations did not indicate a plume of beryllium from the plant operations. The 1989 data were judged more credible than the 1971 data because of better documentation of analytical procedures, more rigorous quality assurance, and improved analytical methods and equipment (*17*).

Site personnel evaluated the distribution of metals in Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) (*18*) Operable Unit (OU) 2 (onsite areas) to investigate the potential contamination of surface soils from windborne dispersal in OU 3 (offsite areas). If contamination of soils onsite was a result of activities at the plant, the soil sampling results were expected to show a distinct spatial trend of decreasing concentrations with increasing distances from areas of the plant where beryllium was used. CERCLA program personnel reasoned that if metal contamination of soil in OU 2 was at background concentrations or appeared to be a result of localized incidents of contamination and no spatial trends could be identified, then contamination in offsite (OU 3) soils was unlikely and sampling of OU 3 soils at distances further out would not be warranted (*18*). Site personnel compared onsite samples with results from two studies of background concentrations: Rock Creek and the Background Soils Characterization Project (*18*). The CERCLA characterization

study found a mean beryllium concentration in OU 2 soils of 0.68 µg/g, a standard deviation of 0.21 µg/g, and a coefficient of variation of 0.31. The beryllium concentrations in OU 2 soils were similar to those for Rock Creek soil samples, which had a mean value of 0.68 µg/g and a maximum concentration of 0.96 µg/g. The Background Soils Characterization Project study (*18*) showed a similar mean of 0.66 µg/g. The U.S. Geological Survey geometric mean for beryllium concentrations in the Front Range soil was 1.2 µg/g (*18*). Spatial trends in the soil data or recognizable plumes were not observed (*14,18*). Taken together, the soil data suggest that beryllium deposited on soil from RFP releases is not detectable.

Ambient air. We reviewed historical ambient air monitoring for beryllium near the RFP. The Dow Chemical Company (Golden, CO) site survey monthly reports from the 1950s contain some qualitative statements and a few quantitative measurements of beryllium in ambient air. Routine monitoring was conducted from 1970 to 1976 and reported in the Dow Chemical Company monthly environmental reports. In the 1970s, the RFP beryllium releases were less than the U.S. Environmental Protection Agency (EPA) discharge limit of 10 g per stationary source for a 24-hr period (*20*).

Monthly average concentrations measured onsite from January 1972 to February 1973 ranged from 0.7 to 1.5 ng/m^3. They were similar to monthly average concentrations measured by offsite samplers over the same period, which ranged from 0.2 to 1.5 ng/m^3. The long-term average beryllium concentration in onsite air from June 1973 to October 1976 was approximately a factor of 3 greater than the median estimated natural background concentration of 0.1 ng/m^3 reported in Rope et al. (*21*). The resuspension of contaminated soil did not appear to contribute significantly to offsite air concentrations (*21*).

Time trend analysis suggests that the concentrations in onsite air appear unrelated to the amount of beryllium released from the plant (*21*). The monitoring data support the atmospheric transport model predictions that offsite air concentrations of beryllium were well below background concentrations.

Beryllium was also present in waste, some of which was discharged into the solar evaporation ponds at the RFP. However, resuspension or leaching of beryllium in waste has not occurred at a level that warrants inclusion in our study.

Health Hazards of Beryllium

To understand the health hazards of beryllium, it is important to review the regulatory standards for beryllium in air, the evidence of carcinogenicity, and the literature on chronic beryllium disease. Because of its use in the nuclear weapons industry, the Atomic Energy Commission recommended occupational and community ambient air standards for beryllium in 1949 (*22*). These standards greatly reduced exposures in and around beryllium plants. The community air standard became the first ambient air quality standard in the United States; it preceded all others by approximately 25 years, and the standard remains unchanged to this day (*23*). The ambient air standard, also called the neighborhood air standard, limits beryllium concentrations in air surrounding factories to 0.01 µg/m^3 averaged over a 30-day period (*24*).

Beryllium carcinogenicity. Numerous studies have shown that beryllium compounds are carcinogenic in experimental animals by several routes of exposure, including inhalation; however, there has been considerable debate as to whether beryllium can cause cancer in humans.

A number of epidemiologic studies have reported an increased risk of lung cancer in beryllium workers, but deficiencies in the studies have not allowed unequivocal conclusions to be made (*25–27*). Criticisms include little or no consideration of smoking history or exposure to other potential lung carcinogens and underestimation of expected cancer deaths in control populations (*25,28*).

In a review of the U.S. beryllium case registry data, Hardy (*29*) reported that there was no evidence to support beryllium as a human carcinogen, but the author recommended workers be studied.

Four epidemiologic studies conducted before 1970 did not clearly demonstrate a relationship between exposure to beryllium compounds and the occurrence of human cancer, but excess risk was suggested by the results of all of the studies (*26,30–32*).

Additional studies in the 1990s found excess risk of lung cancer in workers enrolled in the beryllium case registry (*33*). Occupational exposure to beryllium compounds was the most plausible explanation for the increased risk of lung cancer observed in these studies (*34*).

Four International Agency for Research on Cancer (IARC; Lyon, France) working groups (in 1972, 1980, 1987, and 1993) reviewed the animal and epidemiologic data on beryllium carcinogenicity. The first working group considered the epidemiologic studies available at that time as inadequate to evaluate human carcinogenicity. In 1980 and 1987, the working group concluded that beryllium was carcinogenic to animals, although epidemiologic evidence was limited. The working groups classified beryllium as a suspected human carcinogen. Epidemiologic evidence was again carefully scrutinized by the IARC working group convened in 1993. The proceedings of the 1993 conference (*35*) state that compounds of beryllium are carcinogenic in animals by a number of routes, and several beryllium compounds produce lung tumors in rats exposed by inhalation. The working group concluded that there was sufficient evidence in experimental animals for the carcinogenicity of beryllium and beryllium compounds. After a review of all available epidemiologic studies, the working group concluded that there was also sufficient evidence in humans for the carcinogenicity of beryllium and beryllium compounds. However, controversy about the classification of beryllium as a human carcinogen continued.

Studies implicating beryllium as an occupational carcinogen have examined lung cancer in cohorts exposed in the 1930s and 1940s—before industrial hygiene controls were in place and when concentrations were orders of magnitude higher than permitted today. Statistically significant increases in lung cancer have been difficult to demonstrate in workers exposed to lower levels (*36*).

Currently, beryllium is classified by the EPA as B1, a probable human carcinogen. The weight-of-evidence classification was changed from B2 to B1 in April 1998, but the slope factor remained the same (*32,37*).

Chronic beryllium disease. Chronic beryllium disease is a progressive granulomatous disease. Although the lung is primarily involved, it is a systemic disease and granulomatous inflammation may involve other organs. A delayed hypersensitivity reaction is thought to play a central role in the pathogenesis of chronic beryllium disease. Sensitization to beryllium can be detected by measuring *in vitro* proliferative responses of bronchoalveolar lavage lymphocytes or peripheral blood lymphocytes to beryllium. Clinical and experimental animal data on chronic beryllium disease support an immunologic hypersensitivity mechanism for chronic beryllium disease. Factors that identify immunologic hypersensitivity include the insidious nature of the disease, a long latency between exposure and onset, the granulomatous nature of the lung lesions that develop, berylliosis patients' delayed skin hypersensitivity reactions to beryllium compounds, peripheral blood lymphocytes and bronchoalveolar lymphocytes in people with chronic beryllium disease that undergo blast transformation and release a migration inhibition factor after exposure to beryllium *in vitro*, and the lack of a dose–response relationship (*29,38–41*).

Susceptibility to sensitization is likely to have a genetic basis. Recently, a genetic marker was identified in people with sensitivity to beryllium (*42*). It was concluded

that people with this genetic marker have a significantly increased probability of developing sensitization than those without it (*43*). However, it appears that approximately 30% of the population has the genetic marker and, at most, only about 2–15% of exposed workers become sensitized (*23*).

Most commonly, researchers estimate that 1–5% of beryllium-exposed workers develop chronic beryllium disease (*25,44,45*). Sensitization rates may be higher: Kriess et al. (*46*) reported rates of 2.9–15.8% for beryllium-exposed persons.

Most cases of chronic beryllium disease have occurred in people working in industries processing or using beryllium; however, cases of chronic beryllium disease have been reported in people living near processing plants and in families of beryllium workers, perhaps from exposure to airborne beryllium carried from a plant or from handling contaminated workers' clothing. Chronic beryllium disease has also developed in people in the nonprocessing areas of factories; these people were likely exposed to small amounts of beryllium (*25,29,47,48*). Although Kriess et al. (*46*) reported that the degree of beryllium exposure was associated with disease rates, they found that sensitization occurred in workers with exposures as short as 1 month or in people with unrecognized exposure.

The occurrence of beryllium disease in those with inadvertent or seemingly trivial exposure has been reported in secretaries and security guards at the RFP (*46*) and other facilities (*23*), a janitor in a ceramics company (*23*), and in members of workers' households and neighbors around beryllium extraction plants (*22,32,44*). Cases of chronic beryllium disease that occur in people living in the vicinity of the beryllium plants are termed neighborhood cases (*27*).

In a report summarizing the relationship between the incidence of nonoccupationally related cases of chronic beryllium disease and the levels of atmospheric contamination in the area of a beryllium extraction plant, Eisenbud et al. (*22*) observed that the incidence of disease was a function of the concentration to which the residents were exposed. The incidence of disease within 1/4 mile was approximately 1%, or 5 of 500 people (*22*). The cases of chronic beryllium disease in the 1930s and 1940s in Salem, Massachusetts, occurred almost entirely in fluorescent lamp manufacturing workers. The exceptions were three neighborhood cases: a night watchman, a near neighbor, and a housewife with two young women who were fluorescent lamp workers living in her home. Protection was minimal, and workers were exposed to high levels of beryllium phosphors (*29*).

Chronic beryllium disease was epidemic in the 1940s, leading to the establishment of the beryllium case registry in 1951 (*23,27*). In 1983, Eisenbud and Lisson (*44*) reviewed the beryllium case registry's 224 acute and 622 chronic cases of beryllium disease. These cases included 577 chronic beryllium disease cases due to occupational exposure and 65 cases attributed to ambient air pollution. Forty-two cases were attributed to ambient air exposure in areas near beryllium plants and 23 to exposure to dust brought home on work clothes. They reported no new cases for individuals exposed after 1972 and believed that control measures implemented in the 1950s had reduced chronic beryllium disease despite a marked increase in the use of beryllium (*44*). However, the results of more recent research and clinical work have led to questions about the effectiveness of beryllium control measures and standards on reducing the incidence of chronic beryllium disease. Although many researchers have praised the apparent effectiveness of the air standards for beryllium and have asserted that no new cases of beryllium disease have occurred since the observance of these limits (*49*), others believe that the occupational standards may not be protective for sensitization (*23,43*) and that the limit designed to protect the general public may not be low enough (*41*). The EPA considers the ambient air standard protective for the public with ample margin of safety (*27*).

Evidence exists for biologic responses and possible sensitization occurring after exposure to levels far below the current threshold limit values (*23,41*). In the 1998 EPA toxicological review, EPA researchers stated that several studies observed chronic beryllium disease in people chronically exposed in modern plants that are generally in compliance with the workplace standard for beryllium (the permissible exposure limit) of 2 $\mu g/m^3$ (*32*).

A clear dose–response relationship or duration of exposure–response relationship has not been established for chronic beryllium disease, which is interpreted as involving a delayed hypersensitivity that may be induced by low exposures. Chronic beryllium disease can develop in people with relatively low exposures, whereas nonsensitized people experiencing high exposures may not develop the disease (*30,31,38*). Even slightly exposed individuals, such as the neighborhood cases, sometimes show severe clinical forms of the disease (*22,29*).

Recent studies published by Kriess et al. (*46*) suggest that both individual sensitivity and degree of exposure or exposure circumstances are important in determining the risk of developing chronic beryllium disease. Although chronic beryllium disease cases have been associated with trivial or unrecognized beryllium exposure, chronic beryllium disease rates were higher in workers with presumed greater beryllium exposure, seeming to challenge the no dose–response character for chronic beryllium disease.

In the 1998 reevaluation of beryllium for the Integrated Risk Information System (IRIS), EPA investigators derived a reference concentration (RfC) for beryllium (*37*). The EPA defines the RfC as an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious effects during a lifetime. The EPA reevaluation cited the occupational study done by Kriess et al. (*50*) as the basis for a lowest observable adverse effect level. The effect was beryllium sensitization, measured by the lymphocyte transformation test, which is a very sensitive end point. An RfC of 0.02 $\mu g/m^3$ was determined based on the results of the Kriess et al. (*50*) study. Other worker studies and studies of community residents living near a beryllium plant (*22*) were also considered by the EPA. The uncertainty in the RfC is large. The EPA reevaluation included uncertainty factors for human variability, the less-than-chronic exposure duration in the epidemiologic study used, the sensitive nature of the end point, and the poor quality of the exposure monitoring in the study (*32*).

In general, the most appropriate end point for risk assessment is the effect that occurs at the lowest exposure. Because chronic beryllium disease can develop with low-level exposure, it may be a better end point than lung cancer for assessing risk to low-level exposures. However, chronic beryllium disease may not be dose related, and the percentage of an exposed population that might be expected to develop the disease at a given exposure level is not known (*36*). Future research and studies now in progress may answer the questions of whether a positive lymphocyte transformation test always corresponds to a case of chronic beryllium disease and at what exposure level sensitization occurs.

Methods

We confirmed annual release estimates, release points, and the percentage contribution to the total releases from the site and used them for the calculations (*4*). The greatest release occurred in the year 1968.

For this assessment, inhalation of air is the exposure pathway of concern. Beryllium is not well absorbed after ingestion; is relatively immobile in surface water, tending to absorb to soils and sediments; and would not have been transported offsite in large quantities. Although we could have evaluated beryllium intake from ingesting vegetation subject to deposition from the air, livestock

inhaling air, surface water, soil and sediment, livestock ingesting soil or sediment, vegetation grown in soil, and livestock ingesting vegetation grown in soil, these pathways would be expected to contribute only a small amount to overall risk.

Cancer potency. The EPA weight-of-evidence classification for beryllium is B1, a probable human carcinogen. Probable carcinogens are defined by the EPA as chemicals with sufficient evidence of carcinogenicity in animals with limited human data. The weight-of-evidence classification was changed from B2 to B1 in April 1998 when beryllium was reevaluated for the IRIS database (*37*). A B2 carcinogen is defined by the EPA as having sufficient evidence of carcinogenicity in animals but inadequate evidence or a lack of human data. The reevaluation involved a review of more recent studies, especially the occupational cohort mortality study by Ward et al. (*34*). After considering the available data, EPA investigators recommended that the existing unit risk value, based on a study by Wagoner et al. (*30*), be retained (*37*).

The estimate of the excess lifetime cancer risk is the product of the dose and the carcinogenic potency slope factor (*SF*): excess cancer risk = beryllium exposure concentrations × *SF*.

Cancer *SF*s are usually derived from animal studies using mathematical models (most commonly the linearized multistage model) to estimate the largest possible linear slope (within the 95% confidence limit) at extrapolated low doses that are consistent with the data. The *SF* is expressed in units of the inverse of milligram intake per kilogram body weight per day (kg-day/mg). It represents the 95% upper confidence limit of the probability of a carcinogenic response per daily unit intake of a chemical over 70 years. The *SF* (and risk) is characterized as an upper-bound estimate. The true risk to humans, although not identifiable, is not likely to exceed the upper bound estimate.

The inhalation unit risk factor is the risk per unit concentration in air, calculated by dividing the *SF* by 70 kg and multiplying by the inhalation rate (20 m^3/day) (*51*).

$$UR = \frac{SF \times BR}{BW \times CF} \quad [1]$$

where *UR* = unit risk ($m^3/\mu g$), *SF* = slope factor (kg-day/mg), *BR* = breathing rate (m^3/day), *BW* = body mass (kg), and *CF* = correction from milligrams to micrograms (1×10^3). Using this relationship, we calculated an *SF* of 8.4 kg-day/mg from the mean of the unit risk values published in IRIS (*37*).

For the quantitative estimate of the carcinogenic risk from inhalation exposure, we calculated the inhalation unit risk value using the human occupational epidemiologic data of Wagoner et al. (*30*).

Relative risk estimates were derived from the smoking-adjusted lung cancer death data. The relative risk estimates ranged from 1.36 to 1.44, and the 95% confidence limits of these estimates, 1.98 and 2.09, were used to estimate the lifetime cancer risk. The estimates were based on one dataset using a range of estimated exposures and exposure durations. The effective dose was calculated by adjusting for durations of daily (8 of 24 hr) and annual (240 of 365 days) exposure and the fraction of the lifetime at risk (duration of employment) (*37*). Because of uncertainties in the beryllium exposure levels and exposure times, unit risks were derived using two estimates each of concentration: fraction of lifetime exposed and relative risk. Table 1 summarizes these data. The recommended value for use in risk assessment published in IRIS (*37*) is 2.4×10^{-3}, the arithmetic mean of the eight derived unit risks. The values are conservative, calculated using the 95% confidence limit of the relative risk estimates. Absorption of beryllium is taken into account in the development of unit risk levels. Although based on human data, which generally provide for more confidence than animal data, the quality of the study on which the estimates are based is considered poor because the study was confounded by several variables. A quantitative assessment based on animal studies was reported to have resulted in a similar estimate of risk (*25,37*).

The *Health Assessment Document for Beryllium* (*27*) describes deficiencies of the epidemiologic data, efforts by the EPA Carcinogen Assessment Group to adjust the data for use in calculating cancer potency, and assumptions and models used to extrapolate from high occupational exposures to low-level exposures.

Uncertainties in the slope factors. Slope factors are uncertain. The values used for this assessment are those recommended by the EPA in the IRIS database (*37*). They were derived from a range of epidemiologic data, which are summarized in Table 1. There are obvious limitations to developing values from the results of a single worker epidemiologic study with confounding factors and limitations of its own. Uncertainties associated with the concentrations of beryllium in the workplace, duration of exposure, dosimetry, and other assumptions used in determining the unit risk values were discussed by the EPA (*27*) but were not quantified.

The relative risk estimates were used to provide a probable range and central value rather than just a 95% confidence limit value. The occupational epidemiologic study on which the cancer potency determination was based reported a range for median exposure of 100–1,000 $\mu g/m^3$. Furthermore, an assumption was made that the ratio of exposure duration to years at risk ranged from 0.25 to 1.0. The mean of the potency factors derived using these assumptions was reported in the IRIS database (*37*). The maximum and minimum values (*27*) can be used to calculate a minimum and maximum *SF*. The maximum risk per microgram per cubic meter value of 7.16×10^{-3} corresponds to an *SF* of 25 kg-day/mg, and the minimum risk per microgram per cubic meter value of 1.61×10^{-4} corresponds to an *SF* of 0.56 kg-day/mg. These values were used to approximate an uncertainty distribution for the *SF* assuming a triangular distribution, with the most likely value of 8.4 kg-day/mg.

It is important to note that EPA *SF* values are not used to determine true carcinogenic risk to an individual. Traditionally these values have been used to screen contaminants, determine cleanup levels, or show no impact in prospective assessments. The risk values calculated in this report are not to be interpreted as actual carcinogenic risk to the selected receptors. Rather, the calculated risks represent upper-bound estimates that are not expected to be exceeded for a given intake of beryllium.

Atmospheric transport modeling. The approach taken to calculate atmospheric transport of beryllium involved first estimating an annual average *X/Q* [concentration divided by source term (sec/m^3)] for each receptor in the model domain. Concentrations for specific years of the assessment period were calculated by multiplying the

Table 1. Values from human inhalation occupational exposure studies used to calculate unit risk values.[a]

Workplace beryllium concentration ($\mu g/m^3$)	Fraction of lifetime	Exposure ($\mu g/m^3$)	95% upper bound estimate of relative risk	Unit risk ($m^3/\mu g$)
100	1.0	21.92	1.98	1.61×10^{-3}
100	1.0	21.92	2.09	1.79×10^{-3}
100	0.25	5.48	1.98	6.44×10^{-3}
100	0.25	5.48	2.09	7.16×10^{-3}
1,000	1.0	219.18	1.98	1.61×10^{-4}
1,000	1.0	219.18	2.09	1.79×10^{-4}
1,000	0.25	54.79	1.98	6.44×10^{-4}
1,000	0.25	54.79	2.09	7.16×10^{-4}

[a]Data from the U.S. Environmental Protection Agency (*32,37*).

annual quantity of beryllium released to the atmosphere by the *X/Q* value for a given receptor located in the model domain. We accounted for uncertainties in dispersion estimates through multiplicative correction factors. We then used airborne concentrations along with exposure scenarios and the SFs to calculate risk for selected receptors in the model domain.

Five atmospheric transport models considered for use in this study were evaluated by Rood (*52*). Models included a simple straight-line Gaussian plume model [industrial source complex short term, version 2 (*53*)], a complex terrain model [terrain-responsive atmospheric code (*54*)], and several Gaussian puff models: INPUFF2 (*55*), TRIAD (*56*), and the regional atmospheric transport code for Hanford emission tracking (RATCHET) model (*57*). The results of this evaluation indicated that no one model clearly outperformed the others. However, the puff trajectory models (RATCHET, TRIAD, and INPUFF2) generally had lower variability and higher correlation to observed values compared to the other models. The RATCHET model was chosen for these calculations because it was particularly well suited for long-term annual-average dispersion estimates and it incorporates spatially varying meteorologic and environmental parameters. Features of the RATCHET model are summarized in Table 2.

Model domain and receptor grid. The model domain (Figure 2) encompasses a 2,200-km^2 (850-mile^2) area (50 km north-south by 44 km east-west). The domain extends 28 km south, 12 km west, 22 km north, and 32 km east from the RFP. Most of the Denver metropolitan area and the city of Boulder are included in the domain. The domain was limited in its western extent because few receptors were present and most of the contaminant plumes traveled east and southeast of the plant.

RATCHET uses two modeling grids. Hourly meteorologic records were used to estimate wind speed and direction, stability, and precipitation on the environmental grid and consider surface roughness features. The concentration grid had spacing one-half that of the environmental grid. Ground-level concentrations and deposition were output at each of these grid nodes. The environmental grid was set at 23 nodes east-west and 26 nodes north-south, with a grid spacing of 2,000 m. The concentration grid had 45 nodes east-west and 51 nodes north-south, with a spacing of 1,000 m. Therefore, concentration estimates were made at 45 × 51 = 2,295 receptor locations in the model domain.

Roughness elements (such as trees and buildings) and small-scale topographic features (such as rolling hills) have a frictional effect on the wind speed nearest the surface. The height and spacing of these elements determine the frictional effects on the wind. These effects are directly related to transport and diffusion and affect atmospheric stability, wind profiles, diffusion coefficients, and the mixing-layer depth. The surface roughness length parameter is used to describe these roughness elements and is a characteristic length associated with surface roughness elements. In RATCHET, estimates of the surface roughness length are defined for each node on the environmental grid. In our simulations, we selected a value of 0.6 m to represent residential and urban environs. Farmland, which is predominant in the northeast part of the model domain, was assigned a value of 0.05 m. Range and open land consisting of rolling grass hills were assigned a value of 0.07 m. Nodes that encompass the range and farmland designation were selected based on the topographic contours and land use maps. The foothills and downtown Denver were assigned a value of 2.0 m and open water (Standley Lake) was assigned a value of 0.001 m.

Meteorologic data. Meteorologic data for the operational period of Rocky Flats (1952–1988) are sporadic, incomplete, and of questionable integrity. We initiated an extensive data search in 1994 to locate missing data and interview personnel who were involved with measurements at the site. No new data were recovered, but several personnel reported problems with the recording instrumentation at the RFP, which resulted in the measured wind direction being off by 180°. In 1994, the RFP hired a subcontractor to compile, screen, validate, and analyze historical climatological data (*58*). A draft report was issued in February 1995. It contained monthly and annual summaries of wind speeds, wind directions, precipitation, temperature, and other parameters for the years 1953–1993. Although these data are of interest and may be important for some aspects of modeling, they lacked the resolution required for detailed atmospheric transport modeling. High quality meteorologic data for the RFP are available from 1984 to the present. These data were recorded at the 10-, 25-, and 61-m levels from a 61-m (200-ft) tower located in the southern portion of the RFP industrial area.

It was concluded that meteorologic data taken during the time the RFP was operating were incomplete, unreliable, and unsuitable for atmospheric transport modeling for most of the period from 1952 to 1988. Therefore, a technique that uses surrogate data spanning a different time period and is often utilized in prospective analysis was used to make annual average dispersion estimates for past releases. Federal regulations consider a 5-year database adequate for predicting annual-average air quality impacts at a site. We used meteorologic data spanning a 5-year period (1989–1993) in these simulations; the data were taken at two recording stations located at the RFP and at Denver Stapleton International Airport. The Denver Stapleton International Airport meteorologic station was located 24 km (15 miles) east and 14 km (8.7 miles) south from the center of the RFP. These data included hourly measurements of wind speed, wind direction, cloud cover, and precipitation. Meteorologic conditions in the Denver metropolitan area can differ significantly from those at Rocky Flats (*59*). Therefore, it is unreasonable to use meteorologic data from Denver alone for simulations

Table 2. Features of the RATCHET model.

Feature	Representation in RATCHET
Domain area[a]	2,100 km^2
Node spacing[a]	2,000 m
Source term	Hourly release rates
Meteorologic data	Hourly
Surface roughness	Spatially varying
Wind fields	$1/r^2$ interpolation
Topographical effects	None explicit[b]
Wind profile	Diabatic
Stability	Spatially varying based on wind, cloud cover, and time of day
Precipitation	Spatially varying, three precipitation regimes with different precipitation rate distributions
Mixing layer	Spatially varying, based on calculated values for each meteorologic station
Diffusion coefficients	Based on travel time and turbulence levels
Dry deposition	Calculated using resistance model
Wet deposition	Reversible scavenging of gases, irreversible washout of particles
Model time step	15 min maximum, 15 sec minimum
Output frequency[c]	Daily
Uncertainty	Options available for Monte Carlo simulation within the code

RATCHET, regional atmospheric transport code for Hanford emission tracking.
[a]Modified from the original RATCHET specification for use at Rocky Flats. [b]The model does not account for terrain elevation changes relative to the plume height explicitly. However, topographical influence on the wind field may be accounted for by incorporating multiple meteorologic stations in the model domain. [c]Modified to output annual average concentrations at user specified grid nodes.

involving releases from Rocky Flats. In these simulations, initial plume trajectories were primarily influenced by the wind direction at Rocky Flats. Only after plume elements were transported to the Denver metropolitan area were trajectories and dispersion influenced by meteorologic conditions present there.

Stability classes were calculated separately for the RFP and Denver Stapleton International Airport meteorologic recording stations using the general classification scheme discussed in Pasquill (*60*), Gifford (*61*), and Turner (*62*). This typing scheme uses seven stability categories ranging from A (extremely unstable) to G (extremely stable) and requires estimates of sky cover and ceiling height. We assumed cloud cover and ceiling height data for both stations were the same and obtained the data from the Denver Stapleton International Airport data. We also assumed hourly precipitation records from Denver Stapleton International Airport were consistent over the entire model domain and we segregated the data into integer values as required by RATCHET.

Source characterization. Estimated releases of beryllium to the atmosphere were provided by ChemRisk (*4*). Twenty-five percent of the beryllium released to the atmosphere was attributed to building 444 and 19% was attributed to building 776 (*1*) (Figure 3). Building 444 contained the beryllium foundry where machining, casting, and milling of beryllium occurred. Beryllium milling and machining did not occur in building 776, but some materials containing beryllium were processed. Therefore, beryllium was monitored in the plenum exhaust. Plenum exhaust was passed through HEPA filtration before its release to the atmosphere. The remaining 54% of the atmospheric beryllium releases originated from 11 other buildings surrounding buildings 444 and 776. For these simulations, we assumed that all beryllium originated from buildings 776 and 444. Combined releases were proportioned between the two buildings based on the relative contribution each building had to their combined total. Therefore, the proportion from building 444 was 0.25/(0.25 + 0.19) ≈ 0.6 or 60% and the remainder (40%) was proportioned to building 776 (Table 3). Releases from building 776 reportedly originated from five roof vents. The roof vents were hook-shaped and directed flow down toward the top of the roof. Therefore, the modeled release height was the height of the building. The building height was 11.6 m (38.1 ft) and the horizontal dimensions were 61 × 104 m (200 × 341 ft). The vents were assumed to be distributed across the roof, resulting in an area source geometry. The area source was simulated by modifying the initial diffusion coefficients using a procedure described by Petersen and Lavdas (*55*). The initial horizontal diffusion coefficient (σ_y) is the horizontal dimension of the source divided by 4.3, and the initial vertical diffusion coefficient (σ_z) is the height of the source divided by 2.15. For these simulations we used the 61-m (200-ft) length as the horizontal source dimension.

Releases from building 444 were assumed to have occurred from a point source on the roof [4.5 m (15-ft)] with no buoyant or momentum-driven plume rise. We ignored effects on the initial plume dispersion because of building wakes. At distances of approximately 2 km (1.2 miles), building wake has little effect on measured atmospheric concentrations (*63*). Ramsdell (*64*) showed that for ground-level releases, modeled air concentrations > 1 km (0.6 miles) from the source are relatively unaffected by building wakes. The nearest receptor is > 3 km (1.9 miles) from building 776.

Atmospheric releases of beryllium from building 444 originated from vent 122 after passing through two stages of HEPA filtration (*1*). Effluent containing beryllium reportedly passed through HEPA filtration, resulting in

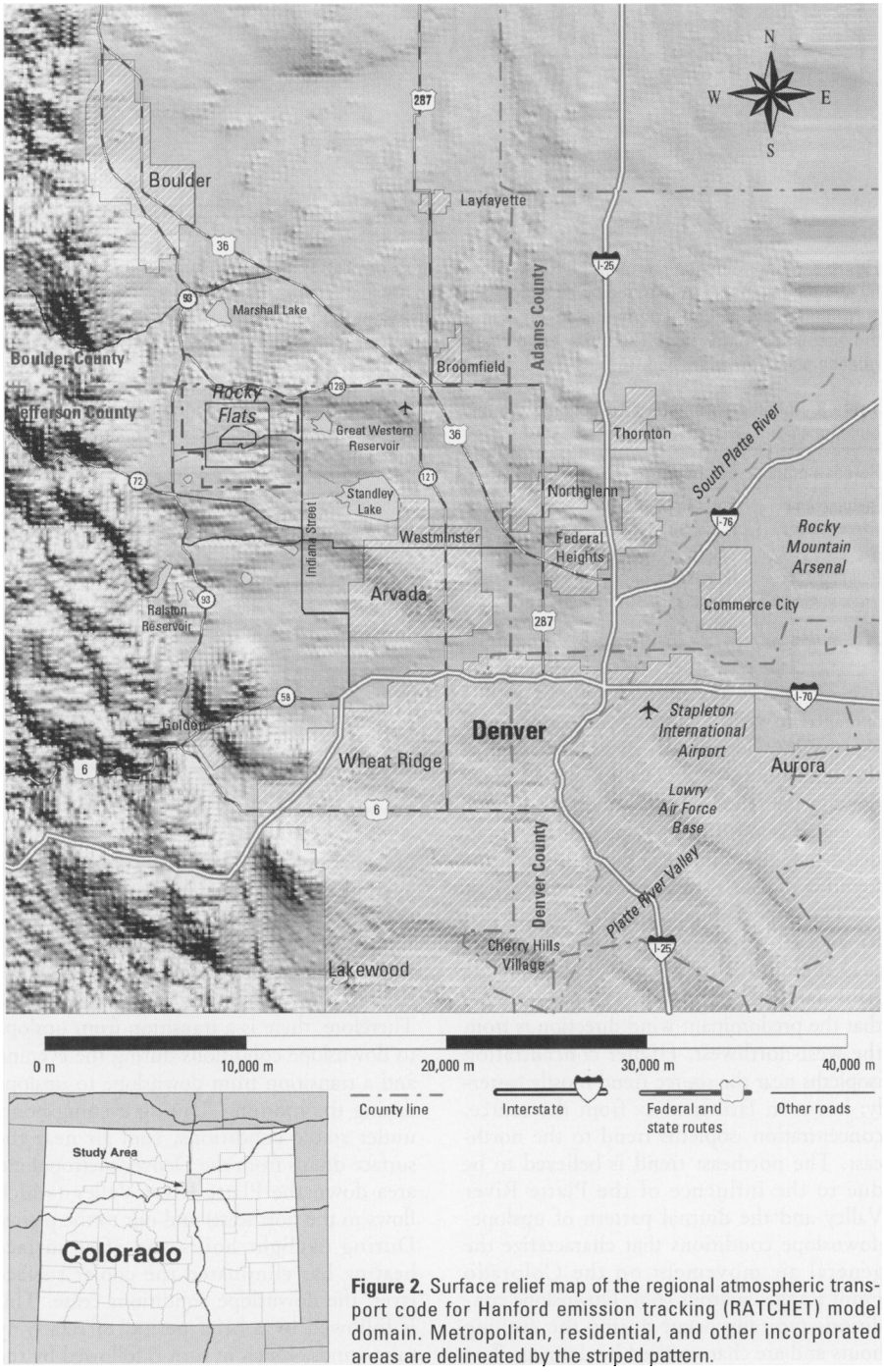

Figure 2. Surface relief map of the regional atmospheric transport code for Hanford emission tracking (RATCHET) model domain. Metropolitan, residential, and other incorporated areas are delineated by the striped pattern.

the release of particles < 1 μm in diameter. Median particle size for plutonium effluent subject to the same HEPA filtration has been estimated as 0.3 μm (*65*). We assumed the beryllium effluent had the same particle size distribution as the plutonium effluent.

Uncertainties associated with the source term estimates were estimated by ChemRisk (*4*) and were used without modification in this analysis. Uncertainty was represented by a multiplicative correction factor. For releases that occurred before 1971, we applied a lognormally distributed correction factor having a geometric mean of 1.9 and a geometric standard deviation of 2.0 to the source term. For releases during 1971 to 1988, we applied a lognormally distributed correction factor having a geometric mean of 1.4 and a geometric standard deviation of 1.9 to the source term.

Prediction uncertainty. We accounted for model prediction uncertainty by using several multiplicative stochastic correction factors in the dispersion estimate, the meteorology, and deposition and plume depletion. Dispersion uncertainty was based on distributions on predicted-to-observed ratios from field tracer experiments using the Gaussian plume and other models including RATCHET. We derived these values from literature reviews and results from studies specific to this project. Meteorologic uncertainty arises because we used 5 years of meteorologic data spanning a recent time period (1989–1993) to define an annual average *X/Q* value that applied to all previous years of the assessment period (1952–1989). This correction factor was derived from studies performed for the Fernald Dosimetry Reconstruction Project (*66*) and comparisons made at Rocky Flats. We calculated deposition and plume depletion uncertainty factors using the Monte Carlo sampling features of RATCHET. All correction factors were distributed lognormally and were combined with the source term uncertainty to yield distributions of predicted concentrations at selected receptor locations. We used Monte Carlo techniques to propagate model prediction uncertainty through to the final risk calculations. Details of how these correction factors were derived are described in McGavran et al. (*2*) and are summarized in Table 4.

Annual average *X/Q* values. We used the RATCHET model coupled with the meteorologic inputs previously described to calculate an annual average *X/Q* for all concentration grid nodes in the model domain. We calculated annual average *X/Q* values separately for releases from building 776 and building 444 and computed the annual average *X/Q* at each of the grid nodes for each year of meteorologic data (1989–1993) for a constant unit release (1 mg/sec) from each building. The

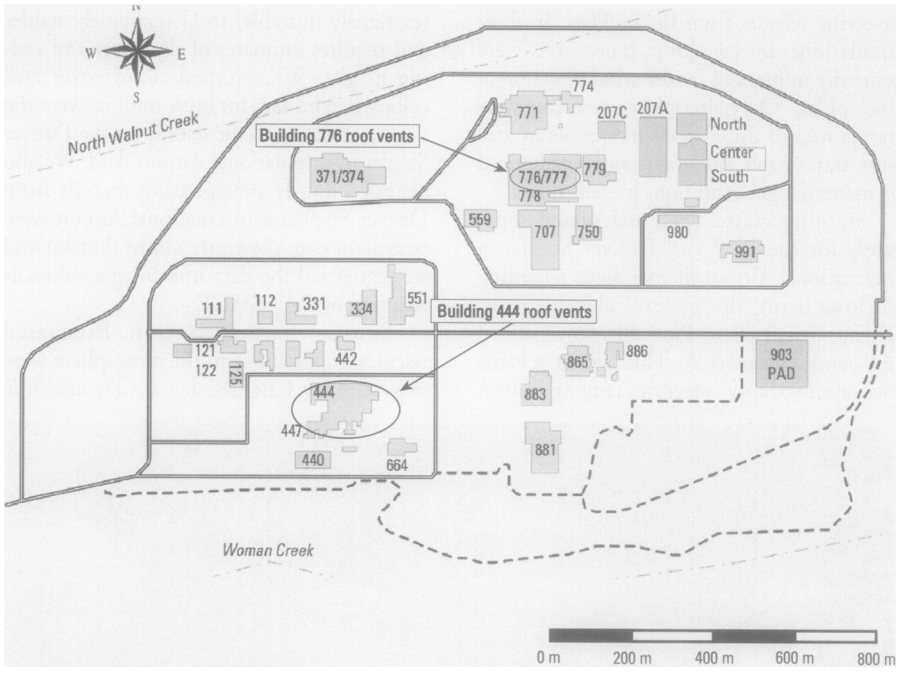

Figure 3. Main production area of the Rocky Flats Plant as of 1990. The buildings were originally identified by two-digit numbers. A third digit was added later. The production area, now called the industrial area, is surrounded by a security perimeter fence. The area between the perimeter fence and Indiana Street to the east is the buffer zone. The buffer zone was expanded to Indiana Street in the 1970s. Major beryllium release points are identified.

Table 3. Release parameters for building 776 and building 444.

Release point	Parameter	Value
Building 444	Release height	5 m
	Diameter of discharge point	2.0 m
Building 776 roof vents	Release height	11.6 m
	Initial σ_r	14.1 m
	Initial σ_v	5.4 m

five *X/Q* values at each grid node were then averaged to yield a 5-year composite annual average *X/Q*. Figure 4 is an isopleth map of the annual average *X/Q* values in the model domain for releases from building 444. We generated isopleth maps using *X/Q* data gridded using the minimum curvature routine found in the Surfer software (*67*).

The dispersion patterns are characterized by an east-northeast trending ellipsoid-shaped plume. Wind roses constructed using RFP data from 1984–1993 (*14*) indicate that the predominant wind direction is from the west-northwest. Higher concentration isopleths near the source trend mostly easterly; however, farther away from the source, concentration isopleths trend to the northeast. The northeast trend is believed to be due to the influence of the Platte River Valley and the diurnal pattern of upslope-downslope conditions that characterize the general air movement on the Colorado Front Range environs (*68*). Downslope conditions typically occur during the evening hours and are characterized by drainage flow

Table 4. Summary of uncertainty correction factors applied to annual average concentration predictions.

Receptor distance (km)	Uncertainty					
	Dispersion		Meteorology		Depletion	
	GM	GSD	GM	GSD	GM	GSD
< 4	1.1	2.2	1.0	1.7	1.0	1.05
8	1.1	2.0	1.0	1.7	1.0	1.09
12	1.1	2.0	1.0	1.7	1.0	1.12
16	1.1	2.0	1.0	1.7	1.0	1.14
20	1.0	2.2	1.0	1.7	1.0	1.16
24	1.0	2.2	1.0	1.7	1.0	1.17
28	1.0	2.2	1.0	1.7	1.0	1.18
> 32	1.0	2.2	1.0	1.7	1.0	1.18

Abbreviations: GM, geometric mean; GSD, geometric standard deviation.

of cooler air from the foothills to the plains. Westerly winds predominate, but the direction may be altered by local topography. Upslope conditions are a result of daytime heating and typically result in easterly winds that prevail during the daylight hours. Therefore, there is a transition from upslope to downslope conditions during the evening and a transition from downslope to upslope during the morning. During evening hours under stable conditions, cool air near the surface drains from the Denver metropolitan area down the Platte River Valley (which flows to the northeast) and out to the plains. During daylight hours and after surface heating has eliminated the cooler surface layer, the downslope conditions cease. This is followed by a brief period of relatively calm winds, which in turn is followed by the

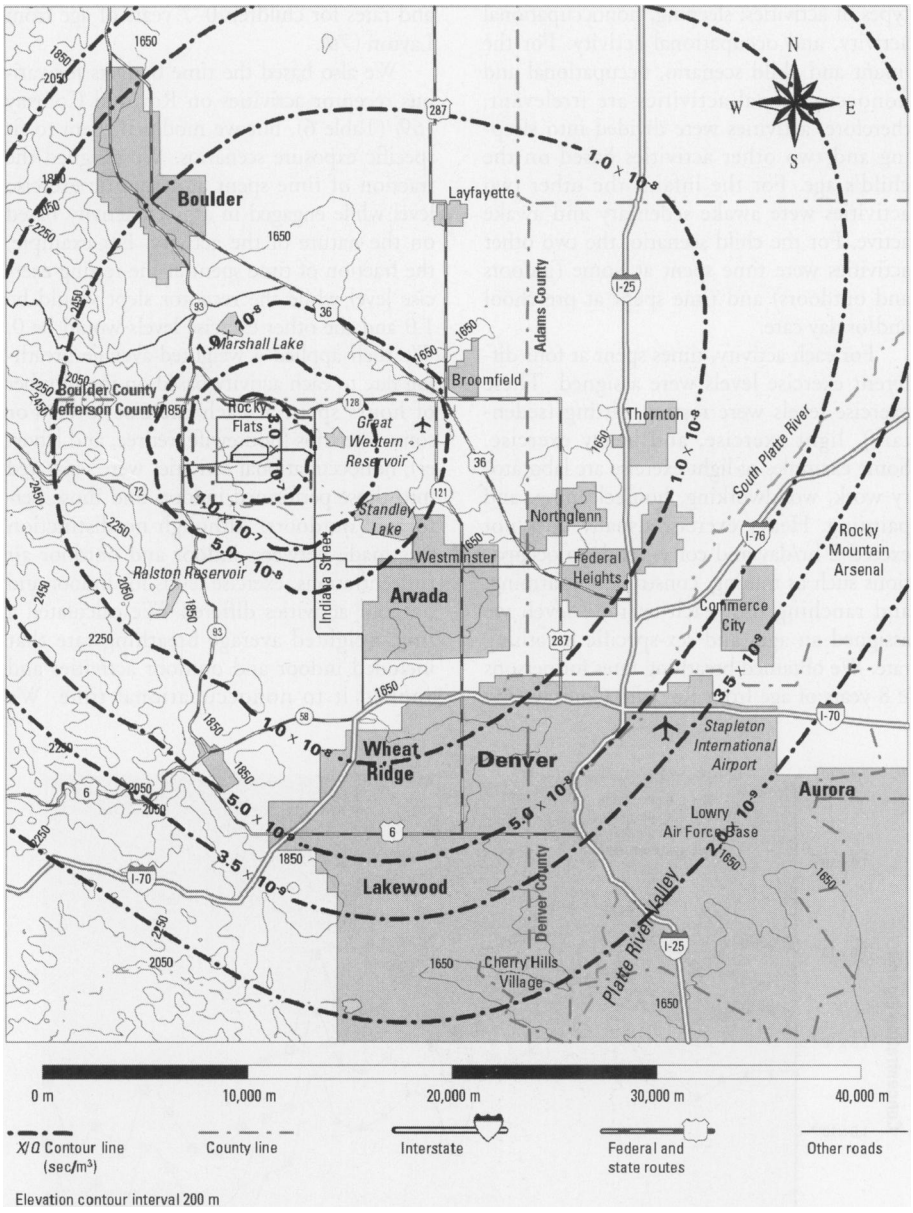

Figure 4. Isopleth map of the annual average *X/Q* for particulate releases from building 444 using meteorologic data from the Rocky Flats Plant and Denver Stapleton Airport from 1989 to 1993.

return of air up the valley or upslope conditions. Meteorologic data at Denver Stapleton International Airport capture these transitions in the Platte River Valley that are reflected in the *X/Q* isopleth map.

Results

Predicted concentrations. We calculated predicted concentrations of beryllium at specific receptors for each year in which source term information was available. Uncertainty in the predicted concentration included uncertainty in the dispersion estimate and source term. The concentration for the i^{th} year is given by

$$C_i = \sum_{j=1}^{2} X/Q_j \times Q_{i,j} \times CF_1 \times CF_2 \times CF_3 \quad [2]$$

where X/Q_j = dispersion factor for source j (concentration divided by source term, year/m^3), $Q_{i,j}$ = annual release of beryllium for the i^{th} year for j^{th} source (building 776 or 444), CF_1 = dispersion uncertainty correction factor, CF_2 = meteorology uncertainty correction factor, and CF_3 = plume depletion uncertainty correction factor.

The correction factors and source term are stochastic quantities; therefore, the concentration is also a stochastic quantity. The concentration to which a hypothetical receptor is exposed is the sum of the prediction concentrations from building 776 and 444 releases. Median value predicted concentrations at the location of highest concentration outside the buffer zone (east of the plant along Indiana Street) for all years in the assessment ranged from 1.3×10^{-6} ng/m^3 in 1986 to 7.3×10^{-4} ng/m^3 in 1968, the year of the highest release (Figure 5). The maximum concentration in the model domain for 1968 was calculated within the plant buffer zone and ranged from 2.5×10^{-3} ng/m^3 (5th percentile) to 6.8×10^{-2} ng/m^3 (95th percentile). This can be compared to an annual average natural background range of 0.03–0.3 ng/m^3 (median of 1×10^{-1} ng/m^3), as estimated in Rope et al. (*20*). Note that the predicted offsite concentrations would be indistinguishable from background concentrations.

We calculated the concentration of beryllium in soil from airborne deposition at the location of highest deposition outside the buffer zone and east of the plant along Indiana Street. We converted integrated surface deposition from 1958 to 1989 to soil concentration by conservatively assuming a sampling depth of 1 cm (0.4 inches) and a bulk density of 1.5 g/cm^3. Predicted soil concentrations ranged from 6.9×10^{-6} (5th percentile) to 2.6×10^{-4} mg/kg (95th percentile), with a median value (50th percentile) of 4.2×10^{-5} mg/kg. These values are well below the mean background soil concentration of 0.66 mg/kg. These calculations support the conclusions of Barrick (*16*) and Allen and Litaor (*19*) that soil concentrations in the vicinity of the plant were not above background and showed no spatial trends or recognizable plumes. We calculated time-integrated concentrations on a receptor-specific basis and integrated concentrations over the duration of time a receptor resided in a given location in the model domain.

Exposure scenarios. One of the key parts of the Rocky Flats dose reconstruction work is calculating health impacts to people living in the surrounding area from materials released during RFP past operations. Dose reconstruction uses a pathways approach to study the potential radiation doses and health risks of past releases on the surrounding communities. The pathways approach begins with learning the types and quantities of materials that were released from a facility and ends with estimating the health impacts which these releases had on the residents in the area. We used mathematical models to model the transport of materials released from the site to the surrounding communities. The following paragraphs describe how we calculated health impacts (lifetime cancer incidence risk) to hypothetical people living offsite from exposure to these releases.

It is not realistic to calculate individual risks for every resident who may have lived or worked in the Rocky Flats area during the plant's operation. Conversely, it is not credible to calculate only a single risk that

would apply to all residents. The risk to which a person is exposed depends on a number of factors, such as lifestyle (did the person spend a great deal of time outdoors or doing heavy work on a farm); when and how long that person lived near the RFP (for example, during the key release events in 1957 and the late 1960s or in the 1970s, when release quantities were lower); age and sex of the person; and where the person lived and worked in relation to the RFP.

To consider these features of a person's life, we developed profiles (or exposure scenarios) for hypothetical but typical residents of the RFP area for which representative risk estimates could be made. Each scenario represented one individual. These scenarios incorporated typical lifestyles, ages, sex, and lengths of time of exposure in the area of the hypothetical residents. These scenarios can help individuals determine risk ranges for themselves by identifying a lifestyle that most closely matches their experience. The scenarios were not designed to include all conceivable lifestyles of residents who lived in this region during the time of the RFP operations. Rather, they provide a range of potential profiles of people in the area.

Risks were calculated from historical beryllium releases from the RFP for nine hypothetical exposure scenarios (Table 5). Inhalation was the only pathway of exposure considered in the assessment. Ingestion of beryllium in water and food and inhalation of deposited beryllium and beryllium attached to soil could have been considered in more detail. However, beryllium compounds are insoluble and tend to adhere to soil, making them relatively immobile and not readily taken up by plants or accumulating in the edible portions of animal products.

Exposure scenarios for the nine hypothetical receptors described in Table 5 were organized according to occupational and nonoccupational activities. Occupational activities included work, school, and extracurricular activities away from the home. Nonoccupational activities included time spent at home doing chores, sleeping, and pursuing leisure activities such as watching television. For some scenarios, the receptor was assumed to perform occupational and nonoccupational activities at a different location. For example, the office worker lives in Broomfield but works in downtown Denver. We also considered the age of the receptor and the years during which exposure occurred when calculating exposures. The last three exposure scenarios represent the same individual at different periods in that individual's life. Cumulative risks over this receptor's lifetime were also calculated.

Breathing rates and time budgets. Each exposure scenario was divided into three types of activities: sleeping, nonoccupational activity, and occupational activity. For the infant and child scenario, occupational and nonoccupational activities are irrelevant; therefore, activities were divided into sleeping and two other activities based on the child's age. For the infant, the other two activities were awake sedentary and awake active. For the child scenario, the two other activities were time spent at home (indoors and outdoors) and time spent at preschool and/or day care.

For each activity, times spent at four different exercise levels were assigned. These exercise levels were resting, sitting (sedentary), light exercise, and heavy exercise. Some examples of light exercise are laboratory work, woodworking, housecleaning, and painting. Heavy exercise usually does not exceed 2 hr/day and corresponds to occupations such as mining, construction, farming, and ranching. For each exercise level, we assigned an age- and sex-specific breathing rate. We obtained breathing rates for persons ≥ 8 years of age from Roy and Courtay (*69*) and rates for children 0–7 years of age from Layton (*70*).

We also based the time budgets for various receptor activities on Roy and Courtay (*69*) (Table 6), but we modified them to fit specific exposure scenarios. We assigned the fraction of time spent at a specific exercise level while engaged in a given activity based on the nature of the activity. For example, the fraction of time spent at the resting exercise level while the receptor slept would be 1.0 and the other exercise levels would be 0. We then applied a weighted-average breathing rate to each activity based on the number of hours spent at each exercise level. For some scenarios (housewife, retiree, and laborer), nonoccupational activities were separated into those performed indoors and those performed outdoors. Although no distinction was made between indoor and outdoor air concentrations, exercise levels for indoor and outdoor activities differed. We calculated a time-weighted average breathing rate that included indoor and outdoor activities and applied it to nonoccupational time. We

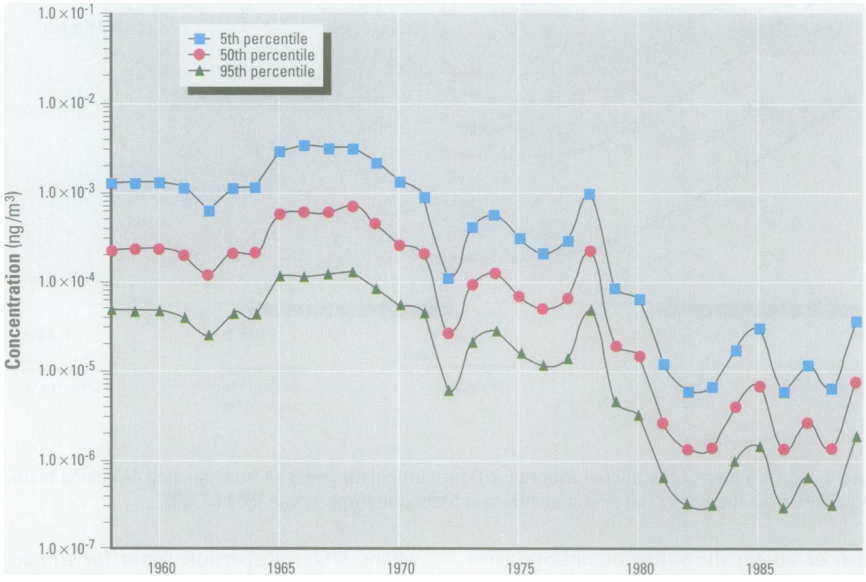

Figure 5. Predicted beryllium concentration as a function of year for a receptor located east of the plant on Indiana Street outside the current buffer zone.

Table 5. Exposure scenario descriptions.

Exposure scenario	Sex	Year of birth	Year beginning exposure	Year ending exposure	Location of occupational activities	Location of nonoccupational activities
Rancher	Male	1925	1953	1989	Indiana Street	Indiana Street
Office worker	Female	1951	1975	1989	Denver	Broomfield
Housewife	Female	1928	1953	1989	Broomfield	Broomfield
Retiree	Male	1923	1978	1989	Arvada	Arvada
Laborer 1	Male	1953	1974	1989	Thornton	Commerce City
Laborer 2	Male	1933	1953	1974	Commerce City	Westminster
Infant[a]	Female	1958	1958	1959	Broomfield	Broomfield
Child[a]	Female	1958	1960	1965	Broomfield	Broomfield
Student[a]	Female	1958	1966	1976	Westminster	Broomfield

[a]These receptors are the same individual. The total risk over her lifetime is also reported.

assumed that each receptor spent 15 days outside the model domain and that contaminant concentrations were the same for indoor and outdoor air.

We calculated time-weighted average breathing rates for the three activities for which each receptor was assumed to be engaged. The time-weighted average breathing rate is given by

$$WBR_j = \sum_{i=1}^{4} BR_i f_{i,j} \quad [3]$$

where WBR_j = time-weighted average breathing rate for the j^{th} activity (m^3/hr), BR_i = breathing rate for the i^{th} exercise level (m^3/hr), and $f_{i,j}$ = fraction of time spent at the i^{th} exercise level for the j^{th} activity.

To reiterate, three activities were defined for each exposure scenario. The location of exposure for occupational activities may be different from nonoccupational activities. The breathing rate during a given activity was the time-weighted average breathing rate of the four exercise levels. Exercise levels were grouped into resting, sitting, light exercise, and heavy exercise.

Calculating risk and uncertainty. Calculating lifetime cancer incidence risk involved three steps: *a*) calculation of the time-integrated concentration at the point of exposure, *b*) calculation of the amount of beryllium inhaled by the receptor, and *c*) multiplication of the beryllium intake by an *SF* that relates the risk of cancer incidence to the amount of beryllium inhaled per day per unit body weight.

In each of these steps, we used Monte Carlo sampling techniques to propagate uncertainty through the calculation. A Monte Carlo calculation consists of multiple iterations or trials of a computational end point (risk). For each trial, parameter values are randomly chosen from distributions that quantitatively describe our knowledge of the parameter. After randomly selecting a set of parameter values, the end point is calculated and the procedure is repeated numerous times until an adequate distribution of the end point is obtained.

Uncertainty in risk estimates was based on uncertainty in the time-integrated concentration and carcinogenic *SF*s. We considered receptor behavior patterns (i.e., the time spent doing different activities at different

Table 6. Time budgets, weighted breathing rates, and body weights[a] for the exposure scenarios.

Scenario	Activity	Fraction of time spent at an exercise level				Hr/day (workweek)	Hr/day (weekend)	Hr/year	Weighted breathing rate (m^3/hr)
		Resting	Sitting	Light	Heavy				
Rancher	Occupational	0.00	0.00	0.25	0.75	8.0	8.0	2,800	2.62
(BW = 78.7 kg)	Nonoccupational	0.00	0.50	0.38	0.13	8.0	8.0	2,800	1.21
	Sleeping	1.00	0.00	0.00	0.00	8.0	8.0	2,800	0.45
Office worker	Occupational	0.00	0.25	0.75	0.00	8.0	0.0	2,000	1.04
(BW = 65.4 kg)	Nonoccupational	0.00	0.50	0.38	0.13	8.0	16.0	3,600	1.00
	Sleeping	1.00	0.00	0.00	0.00	8.0	8.0	2,800	0.32
Housewife	Occupational	0.00	0.13	0.75	0.13	8.0	8.0	2,800	1.33
(BW = 65.4 kg)	Nonoccupational								
	Indoor	0.00	0.50	0.38	0.13	4.0	4.0	1,400	1.00
	Outdoor	0.00	0.38	0.50	0.13	4.0	4.0	1,400	1.11
	Total nonoccupational	0.00	0.44	0.44	0.13	8.0	8.0	2,800	1.06
	Sleeping	1.00	0.00	0.00	0.00	8.0	8.0	2,800	0.32
Retiree	Occupational	0.00	0.50	0.50	0.00	8.0	8.0	2,800	1.02
(BW = 78.7 kg)	Nonoccupational								
	Indoor	0.00	0.50	0.38	0.13	6.0	6.0	2,100	1.21
	Outdoor	0.00	0.50	0.38	0.13	2.0	2.0	700	1.21
	Total nonoccupational	0.00	0.50	0.38	0.13	–	–	2,800	1.21
	Sleeping	1.00	0.00	0.00	0.00	8.0	8.0	2,800	0.45
Laborer 1	Occupational	0.00	0.13	0.50	0.38	8.0	0.0	2,000	1.94
(BW = 78.7 kg)	Nonoccupational								
	Indoor	0.00	0.50	0.38	0.13	6.0	8.0	2,300	1.21
	Outdoor	0.00	0.50	0.25	0.25	2.0	8.0	1,300	1.40
	Total nonoccupational	0.00	0.50	0.31	0.19	–	–	3,600	1.28
	Sleeping	1.00	0.00	0.00	0.00	8.0	8.0	2,800	0.45
Laborer 2	Occupational	0.00	0.13	0.50	0.38	8.0	0.0	2,000	1.94
(BW = 78.7 kg)	Nonoccupational								
	Indoor	0.00	0.50	0.38	0.13	6.0	8.0	2,300	1.21
	Outdoor	0.00	0.50	0.25	0.25	2.0	8.0	1,300	1.40
	Total nonoccupational	0.00	0.50	0.31	0.19	–	–	3,600	1.28
	Sleeping	1.00	0.00	0.00	0.00	8.0	8.0	2,800	0.45
Infant	Awake (sedentary)	0.00	0.71	0.14	0.14	7.0	7.0	2,450	0.33
(BW = 9.4 kg)	Awake (active)	0.00	0.00	1.00	0.00	1.0	1.0	350	0.45
	Sleeping	1.00	0.00	0.00	0.00	16.0	16.0	5,600	0.14
Child	Home								
(BW = 15.8 kg)	Indoor	0.00	0.50	0.42	0.08	6.0	6.0	2,100	0.55
	Outdoor	0.00	0.00	0.67	0.33	1.5	1.5	525	1.04
	Total home	–	–	–	–	7.5	7.5	2,625	0.65
	School (indoor)	0.00	0.80	0.20	0.00	2.5	2.5	875	0.35
	Sleeping	1.00	0.00	0.00	0.00	14.0	14.0	4,900	0.23
Student	Home								
(BW = 44.4 kg)	Indoor	0.00	0.44	0.56	0.00	4.5	8.0	1,925	0.83
	Outdoor	0.00	0.00	0.25	0.75	2.5	6.0	1,225	1.98
	Total home	0.00	0.22	0.40	0.38	7.0	14.0	3,150	1.28
	School								
	Indoor	0.00	0.75	0.25	0.00	6.0	0.0	1,500	0.59
	Outdoor	0.00	0.00	0.25	0.75	1.0	0.0	250	1.98
	Total school	0.00	0.38	0.25	0.38	7.0	0.0	1,750	0.79
	Sleeping	1.00	0.00	0.00	0.00	10.0	10.0	3,500	0.33

BW, body weight.
[a]Body weights were obtained from Finley et al. (*73*).

exertion levels) and their physical attributes (body weight and breathing rate) fixed quantities. We established the exposure scenarios to evaluate risks for hypothetical individuals and did not consider variability within the population of potential receptors.

The procedure outlined above requires an estimate of the time-integrated concentration at the point of exposure. A receptor can be exposed at two locations: place of work (occupational) and place of residence (nonoccupational and sleeping). Consider a Monte Carlo calculation consisting of m trials. The time-integrated concentration of the k^{th} trial ($0 < k \leq m$) for source j and location i is

$$TIC_{i,j} = CF_1 \sum_{l=1}^{n} CF_2 CF_3 X/Q_{i,j} Q_{j,l} \Delta t \qquad [4]$$

where $X/Q_{i,j}$ = dispersion factor for source j and location i (year/m^3), $Q_{j,l}$ =source term for year l and source j (mg/year), CF_1 = stochastic correction factor for dispersion (unitless), CF_2 = stochastic correction factor meteorology (unitless), CF_3 = stochastic correction factor for deposition and plume depletion (unitless), n = number of years exposed, and Δt = time increment (1 year).

Notice that the dispersion correction factor (CF_1) is outside the summation symbol. For each Monte Carlo trial, CF_1 is sampled once, but the correction factors, CF_2, CF_3, and source term are sampled n times. We used this sampling scheme to allow for year-to-year correlation in annual dispersion estimates as discussed earlier. The amount of beryllium inhaled by a receptor for the k^{th} Monte Carlo trial is

$$I = \sum_{j=1}^{2} \left(TIC_{1,j} WBR_1 T_1 + TIC_{2,j} WBR_2 T_2 + TIC_{2,j} WBR_3 T_3 \right) \qquad [5]$$

where I = intake of beryllium by the receptor for the exposure period (mg), $TIC_{1,2,j}$ = time-integrated concentration for occupational and nonoccupational (including sleeping) locations and j^{th} source (mg-year/m^3), $WBR_{1,2,3}$ = time-weighted average breathing rate for occupational, nonoccupational, and sleeping activity (m^3/hr), and $T_{1,2,3}$ = hours per year for occupational, nonoccupational, and sleeping activity (hr/year).

The subscripts 1, 2, and 3 refer to occupational, nonoccupational, and sleeping activity, respectively. The time-integrated concentration values (Table 7) are only calculated at two locations, and the same time-integrated concentration value is applied to sleeping and nonoccupational awake activities. Distributions of time-integrated concentration values in Table 7 are described in terms of their geometric mean and geometric standard deviation. Analyses of the data points in these distributions show that they are best represented by a lognormal distribution. However, in practice, calculations are performed using the actual distribution (made up of m number of trials) and not the lognormal representation. Magnitude of the time-integrated concentration was dependent on the length of exposure, location of exposure, and magnitude of source during exposure. Differences in the geometric standard deviation values between scenarios are mainly related to the length of exposure and magnitude of the dispersion correction factor. Longer integration time typically corresponds to lower geometric standard deviations (but not lower variance) because summing the independent stochastic variables (CF_2 and CF_3) over the integration period results in a lower coefficient of variation of the sum as compared to the coefficient of variation of individual years. The coefficient of variation is the standard deviation of the sum divided by the mean of the sum (σ/μ). Like the coefficient of variation, the geometric standard deviation is a relative measure of the spread of the data within the distribution. The decrease in the geometric standard deviation for longer averaging times is because the relative variability in the time-integrated concentration decreases with increasing integration time.

Finally, calculating the incremental lifetime cancer incidence risk requires estimates of the *SF*. Distributions of *SF*s were described previously in this article. Carcinogenic risk from beryllium inhalation was calculated using the standard risk equations described by the EPA (*71*) and given by

$$R = \frac{SF \times I}{BW \times AT} \qquad [6]$$

where R = cancer incidence risk, SF = carcinogenic slope factor (kg-day/mg), I = distribution of integrated contaminant intake (mg), BW = body mass (kg), and AT = averaging time (70 years × 365 days/year).

Age-specific body weights used in Equation 6 are presented in Table 6. We performed Monte Carlo sampling. Each step of the Monte Carlo simulation is described below.

First, the distributions of time-integrated concentration values (Equation 4) for each receptor activity and each source were calculated first. Nonoccupational and sleeping activities were assumed to occur at the same location. Therefore, two time-integrated concentration values were calculated for each receptor and each source, one for occupational activities and one for nonoccupational and sleeping activities. Each time-integrated concentration distribution contained m number of individual trials. If occupational and nonoccupational activities occurred at the same location, then a single time-integrated concentration value was used for each source.

Second, each of the time-integrated concentration trials is multiplied by the WBR_i and T_i (corresponding to the i^{th} receptor activity), then summed over all sources and receptor activities to yield the total contaminant intake of the k^{th} trial (Equation 5). The procedure was repeated for all m trials.

Third, each estimate of total contaminant intake was multiplied by a randomly selected SF value and divided by body weight and averaging time to give an estimate of the lifetime cancer incidence risk. This calculation was repeated m times to yield a distribution of lifetime cancer incidence risks.

Fourth, percentiles, geometric mean, and geometric standard deviation values were

Table 7. Time-integrated concentrations for each receptor scenario and source for occupational and nonoccupational activities.

Scenario	Activity	Time-integrated concentration, building 444[a] (mg-year/m^3)	Time-integrated concentration, building 776[a] (mg-year/m^3)
Rancher	Occupational	3.5×10^{-9} (2.3)	3.2×10^{-9} (2.3)
	Nonoccupational	3.5×10^{-9} (2.3)	3.2×10^{-9} (2.3)
Office worker	Occupational	3.8×10^{-12} (2.4)	2.5×10^{-12} (2.4)
	Nonoccupational	5.5×10^{-11} (2.1)	4.2×10^{-11} (2.1)
Housewife	Occupational	7.3×10^{-10} (2.1)	5.6×10^{-10} (2.1)
	Nonoccupational	7.3×10^{-10} (2.1)	5.6×10^{-10} (2.1)
Retiree	Occupational	1.2×10^{-11} (2.5)	7.2×10^{-12} (2.5)
	Nonoccupational	1.2×10^{-11} (2.5)	7.2×10^{-12} (2.5)
Laborer 1	Occupational	2.0×10^{-11} (2.2)	1.5×10^{-11} (2.2)
	Nonoccupational	7.8×10^{-11} (2.3)	5.1×10^{-12} (2.3)
Laborer 2	Occupational	8.1×10^{-11} (2.3)	5.4×10^{-11} (2.3)
	Nonoccupational	6.1×10^{-10} (2.1)	4.0×10^{-10} (2.1)
Infant	Occupational	5.5×10^{-11} (2.5)	4.3×10^{-11} (2.6)
	Nonoccupational	5.5×10^{-11} (2.5)	4.3×10^{-11} (2.6)
Child	Occupational	2.0×10^{-10} (2.3)	1.5×10^{-10} (2.2)
	Nonoccupational	2.0×10^{-10} (2.3)	1.5×10^{-10} (2.2)
Student	Occupational	3.4×10^{-10} (2.2)	2.3×10^{-10} (2.2)
	Nonoccupational	4.0×10^{-10} (2.2)	3.1×10^{-10} (2.2)

[a]Geometric mean (geometric standard deviation).

then calculated from the distribution of m risk values.

We calculated the total risk over the lifetime of the individual that represents the infant, child, and student scenarios differently. For each trial, contaminant dose (intake divided by body weight, in milligrams per kilogram) was calculated for each year the receptor was exposed. Note that body weight and breathing rate change as the individual matures. Meteorologic, deposition, and source term uncertainty were applied to each year's dose estimate. The dose was summed across all years of exposure, then multiplied by the dispersion correction factor and slope factor and divided by the averaging time. This process was repeated m times, resulting in a distribution of lifetime cancer risk estimates to the individual.

We adapted FORTRAN routines for generating random numbers and selecting values from normal, lognormal, triangular, and uniform distributions from Press et al. (*72*). The output distributions provided in this article were generated from 2,000 trials.

Risk estimates. The lifetime cancer incidence risks reported here represent the precision in the models and methodology used in the calculation. They should not be used to determine the probability that a real individual within the population will develop cancer. Geometric mean incremental lifetime cancer incidence risk estimates for beryllium inhalation (Table 8) were highest for the rancher scenario (3.9×10^{-10}) and lowest for the retiree scenario (7.5×10^{-13}). The 5th and 95th percentile values of the risk estimates are illustrated in Figure 6. The range of values shown represent the 5th and 95th percentiles on the cumulative density function.

Using the rancher scenario as an example, these risks may be interpreted as follows:

- There is a 90% probability that the incremental lifetime cancer incidence risk to the rancher was between 7.5×10^{-11} (5% value) and 1.8×10^{-9} (95% value)
- There is a 5% probability that the incremental lifetime cancer incidence risk for the rancher was > 8.4×10^{-9} (100% value)
- There is also a 5% probability that the incremental lifetime cancer risk for the rancher was < 7.5×10^{-11}.

Estimated risks were a function of exposure time, exposure duration, and location of exposure. Risk is inversely proportional to body weight, which explains the relatively high risk for the infant scenario. We did not consider age and sex dependencies; furthermore, few data exist to develop such values. Therefore, the risks presented here for the infant, child, and student scenarios must be interpreted with caution because *SF*s for adults were used to compute carcinogenic risk.

Risk estimates are well below the EPA point of departure for acceptable risks (10^{-6} to 10^{-4}). As stated previously, the EPA *SF* values were not intended to represent the true carcinogenic risk to an individual, but were designed to be protective of human health. The risk values reported here, therefore, should be evaluated in light of the EPA point of departure for acceptable risk.

The rancher scenario represents the maximum exposed individual in the model domain because the rancher was placed at the point of highest concentration outside the RFP buffer zone and remained there for the entire operating period of the plant. However, it is recognized that in the past, ranchers could have had cattle grazing within the current buffer zone. There were also bunkhouses or some type of permanent overnight ranch camps to the northeast within the buffer zone. To increase the risk substantially from our estimates, the concentration within the buffer zone would have had to be several orders of magnitude greater than outside the buffer zone. However, this is not the case, as evidenced by the *X/Q* data presented in Figure 4 and differences between the predicted concentration at Indiana Street and the maximum concentration in the model domain. The resulting risk, accounting for occupancy time while exposed to concentrations within the buffer zone, would still be at or below the EPA point of departure for acceptable risk of 10^{-6} to 10^{-4}.

Although beryllium exposures for workers at the RFP have been of great concern and the attention to workers may have caused public concern about health effects due to beryllium exposure offsite, the results of this assessment predicted that lung cancer risk from beryllium exposures offsite was negligible. The risk for chronic beryllium disease in the offsite public is uncertain. The maximum concentration estimated in the entire model domain occurred onsite and ranged from 2.5×10^{-6} (5% value) to 6.8×10^{-5} μg/m^3 (95% value). These concentrations were approximately 300 times less than the EPA RfC of 2.0×10^{-2} μg/m^3. The maximum concentration predicted along Indiana Avenue ranged from 9.4×10^{-7} to 1.4×10^{-5} μg/m^3, concentrations more than 1,400 times less than the RfC. A hazard index calculated using these values would be well below 1. However, because of the complexity and apparent immunologic nature of chronic beryllium disease, it is difficult to conclude that no cases of chronic beryllium disease may have occurred from offsite exposure.

Table 8. Incremental lifetime carcinogenic incidence risk from beryllium inhalation calculated for nine exposure scenarios.

Scenario	GM	GSD
Rancher	3.9×10^{-10}	2.7
Office worker	2.8×10^{-12}	2.5
Housewife	6.3×10^{-11}	2.5
Retiree	7.5×10^{-13}	2.8
Laborer 1	1.1×10^{-12}	2.6
Laborer 2	3.4×10^{-11}	2.5
Infant	7.6×10^{-12}	2.8
Child	2.9×10^{-11}	2.6
Student	4.1×10^{-11}	2.6
Total (child)[a]	7.6×10^{-11}	2.6

Abbreviations: GM, geometric mean; GSD, geometric standard deviation.
[a]Total (child) represents the integrated risk for the infant, child, and student scenarios.

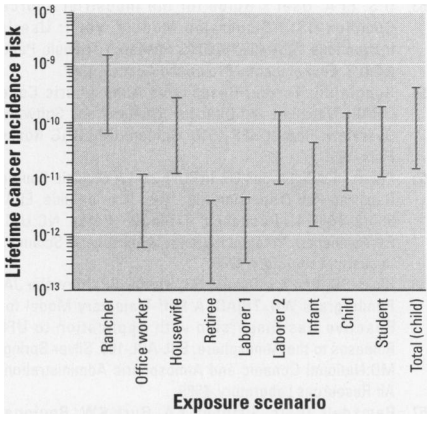

Figure 6. Incremental lifetime cancer incidence risk estimates for the nine exposure scenarios. The total (child) represents the sum of the infant, child, and student scenarios.

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